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THE ACTION OF

INFLUENZA POISON ON THE HEART

AND A STUDY OF

INFLUENZAL ANGINA PECTORIS.

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THE ACTION OF INFLUENZA POISON ON THE HEART, AND A STUDY OF INFLUENZAL ANGINA PECTORIS.

THE former paper on this subject, read before the Philadelphia County Medical Society, was of necessity somewhat crude and hasty, but the subject demands far more attention and careful study, and this must be our excuse for again bringing it before the medical profession.

What is the *cause* of the peculiar condition of the heart during and following influenza? and, as a basis for this inquiry, What is the *condition* of the heart itself? In the answer to these questions necessarily lies the key-note to all treatment. Briefly, we may say, that the heart condition is evidently not due to anæmia and a consequent weakened condition of the heart wall. This is shown by the rapid onset of the symptoms, and their frequent rapid subsidence. It is not inflammatory, for inflammations of the endocardium were exceeding rare, and old endocardial trouble was not prone during the epidemic to be aggravated or lighted up afresh, and subnormal temperature generally existed; even articular rheumatism associated with influenza had little tendency to exo- or endo-cardial mischief. Possibly in some protracted cases the long-delayed fatal event may have been due to nutritional changes in the heart-muscle, consequent, as we shall see later, to continued faulty innervation.

How inextricably influenza was bound up with heart symptoms, heart weakness, and general as well as circulatory depression, the careful observer cannot fail to see. All cases of acute influenza were followed by characteristic nervous depression, sometimes even amounting to a partial paralysis of the muscles in the neighborhood of the catarrhal trouble; some exhibited this depression to a remarkable extent,—not alone in its intensity but in its duration. Whenever and wherever the local process could be detected, depression followed. Where the local catarrh (influenza) was in its commonest and lightest form (naso-pharynx and upper air-passage), there the general depression was most easily borne and most evanescent. (The duration and intensity of the fever accompanying this local catarrh were in some measure indicative of the amount of exhaustion to be expected.) Since all the catarrhs of the peculiar character termed influenzal were followed in this way, the depression would necessarily seem to be consequent, and produced not by the inflammatory process but by some one or more of its products. And without assuming the bacteriology of influenza to be yet perfectly established, we cannot be far from the actual facts of the case if we believe

that a minute growth, animal or vegetable, is the morbific agent in this catarrh, and the only remaining point is to prove, if possible by actual cases and their investigation, how and where this agent acts in producing depression and exhaustion.

Localized simple influenzas of the commonest variety, coming on suddenly and disappearing in a few hours or days, were habitually followed by general nervous symptoms of short duration. Hence in these cases of rapid recovery we need not suppose the system to be germinally invaded any farther than the naso-pharynx and larger bronchi; and the toxic product, if any, formed by such invasion must be responsible for the toxic symptoms. Where other localized catarrhs occurred primarily, as hepatic, gastric, or intestinal, the depression was apt to be more lasting, and where the nervous system seemed to succumb primarily, without any manifest catarrhal process, are we to assume a latent one, perhaps in the gastro-intestinal mucous membrane (spleen), or are we to believe that the nerve-centres and nerve-trunks were themselves invaded, and, if so, by what? The actual colonies of microbic germs or their toxic consequent?

Recently-reported autopsies seem to establish the fact that, in the spinal cord at least, actual lesion of a serious character may exist, and if in the spinal cord, of course in any centre, cerebro-spinal or sympathetic, and in any nerve-trunk at any portion of its course.

Superficially this view of the case seems proved by the frequent occurrence of neuritis after influenza; where the progress of the morbid condition can be clinically watched, at its onset centrally, as shown by shifting from side to side in abortive attacks of pain, showing derangement of the nerve-centre, and in its progress, as indicated by pain on pressure, the painful point progressing from centre to periphery,—as, for instance, in the brachial,—where often both radial and ulnar distribution are affected, until, as the painful point descends below the elbow, one alone is implicated, and in its distribution points back by pain and numbness to the affected nerve.

Internally the problem (in the absence of post-mortem inquiry) is both simpler and more complex. The pneumogastric with its various distributions will account, if we consider it as implicated, for many of the internal manifestations as evidenced in the varying intensity of the lung attacks, at first non-inflammatory, then passing into catarrhal pneumonia or catarrhal phthisis; but this nerve is beyond mediate or immediate touch, and the only way to arrive at conclusions is by comparison, analysis, and reasoning upon the cases brought to our notice. It may be remarked, however, that implication of the pneumogastric will not, as some have supposed, account for all the variety of influenzal manifestations, and while we are forced to the conclusion that the symptoms observed during the disease, and following it, were entirely of a nervous character, the questions still remain: What, then, was the nature of the pathological condition? Was it toxic or morbific, and what systems of nerves were affected? Cerebro-spinal, sympathetic, or both; sensory or motor or both? To discover anything of value we must go over

once more the symptomatology of influenza, and especially of the cardiac cases of the disease, with which we are at present concerned.

Following the first explosive epidemic (1889–90), we had many suddenly fatal and very marked cases of heart-failure, more so than at any other period of the epidemic. In the spring and fall of 1890 there were a number of cases with more chronic symptoms of the disease that finally developed heart-failure of a less intense character, the onset and fatal result being separated by a longer interval. These cases ceased to be met with late in the fall of 1890, nor were they noticed to any extent in the succeeding winter and spring.

The outbreak of December, 1891, brought back, however, the same kind of cases, milder in form, for a couple of months, but not to the same extent, as the former outbreak in 1889. The symptoms in the old varied from those in the young throughout the epidemic. In the old, intermittent heart was very common and was often associated with irregularity. In the young, there seemed to be rather a simple heart weakness.

In the first epidemic blue lips were not infrequent in old and young; in the last outbreak this symptom was seldom met at any age. The heart was not often rapid in its action even when seriously affected, either in young or old; but a few cases of rapid heart were seen, while the vast majority of cases presented slow action,—less slow in the young and vigorous,—sixty-five to eighty beats,—more marked in the aged and weak, often as low as forty-five per minute. In the last year many cases had symptoms of syncope, even complete syncope, sometimes associated with symptoms of heart weakness, which were provoked by slight causes.

Before considering the nervous supply of the heart and the angina pectoris, or anginose cases, we give a few typical cases of simple heart-failure, as commonly met with. Just such histories as these could be given in great numbers, but a few will suffice.

SIMPLE HEART-FAILURE.

CASE I.—Mrs. C., aged forty-three, grippe symptoms for a month, with gradual improvement; on undertaking some work sudden heart-failure followed, attended with profuse sweating, syncope, and after this some blueness of the lips and skin. The heart was irregular, weak, and not accelerated materially. The next day, on rising from bed, she had immediately a recurrence, and subsequently three more, each lighter than the preceding. She had with this condition, at the upper border of the third rib immediately above the left nipple, a tender point on percussion and pressure.

CASE II.—Mrs. C., aged seventy, had apparently recovered from the influenza about two weeks, when, after a long and hurried walk, she was seized with oppression, distress, and a sense of weakness; pulse at the wrist weak but regular; heart beat fifty-four; respirations nearly normal; profuse perspiration. The first attack lasted four hours; the next day she had two attacks, each brought on by attempting to rise, and on each succeeding day for over a week one or two, gradually becoming much lighter, until at last they occurred only in the morning when sitting up to eat. Complete recovery.

LONG-CONTINUED INTERMITTENT HEART.

CASE III.—F. P., aged twenty-five, was left with intermittent heart, more or less persistent; could be brought on by physical exertion, menstrual period, excitement, or by eating. This condition had lasted a whole year; when under treatment by cactus grandiflora, alcohol, and caffeine, with quinine and strychnine, rest and care, and later on with hypophosphites, symptoms finally yielded.

CASE IV.—Dr. L. had grippe in the winter of 1892. This was followed for six months by palpitation and dyspnœa; pulse, while at rest, 52; on slight exertion, 92.

PERSISTENT SLOW HEART.

CASE V.—J. R., aged sixty-two; had attack of grippe in January, 1892; two weeks in bed; had nausea, dizziness, and palpitation. The first day he attempted to resume business, his pulse, when seen shortly after, 58 to 60 (previous pulse when in health, 75 to 80), and while apparently in good health and going about it remains at 60 up to June (five months).

CASE VI.—M. L. W., aged fifteen; attack followed shortly after apparently rapid recovery from the laryngeal form of influenza; began as sudden weakness, while walking to school; became more marked for several mornings, and she was finally sent to bed; prostration very great for a week; pulse under 60, rising on slight exertion to 100 or even 120; complete but slow recovery, with persistent numbness of left arm.

We now come to a few cases illustrative of the rapid alternation of heart symptoms with other conditions.

MIGRATORY AND INTERCHANGING HEART SYMPTOMS.

CASE VII.—Mrs. K. C., aged sixty-four; grippe-lung with marked hæmoptysis; sudden subsidence of physical signs; heart-failure with *rapid* beat, and this suddenly replaced by *delirium*, and early recovery.

CASE VIII.—A. B., grippe; catarrhal pneumonia; heart-failure (rapid heart); enteralgia, and lastly gastric catarrh.

CASE IX.—E. K., aged sixty-eight; grippe; paralysis of deglutition; acute delirium completely disappearing; heart-failure; pulmonary œdema, with blue lips and slow pulse, and death in three hours from appearance of heart symptoms.

CASE X.—T. P. C., aged thirty-four; grippe for nine days; fifth day after, heart became irregular; temperature 103; pulse dropped every third beat; no pain nor uneasy sensation about the heart; impulse regular but weak; excessively nervous, with face more pale than usual. Three days later, still missing every third beat; two days later, every eighteenth to twenty-fourth beat; excessively slow recovery of strength. Pulse not accelerated; no sensation of palpitation. The heart in this case was simply intermittent or weak.

CASE XI.—N. M. C., aged twenty-four; nurse; had severe influenza December 3, 1889. Subnormal temperature and vomiting for the first four weeks; after the first two days after partial recovery and return to nursing, she had pallor, nervousness, faintness on exertion. Left arm and both legs swollen, with marked blueness and red mottling, increased by cold or fatigue, resembling the milder form of Reynaud's disease. This discoloration improved under treatment,—hypophosphites, stimulants (alcohol), and rest.

RÉSUMÉ OF THIS GROUP OF CASES.

The youngest case was fifteen years old (one case, not included in above list, of five years, was met with), the oldest seventy-eight. Most were past

middle life. The pulse was generally slow while at rest, but was hurried by slight exertion,—eating, especially breakfast, was a frequent excitant. Temperatures were generally subnormal, 97° to 97.5°, with a few exceptions. In rare cases there was venous congestion, but generally not; generally it seemed only a change in the rapidity of heart action without any in muscular force. The heart was regular and slow; regular and fast; intermittent, neuralgic, irregular, and slow, and in these latter the absence of bluing of the surface would seem to indicate that the heart was able thoroughly to carry on the circulation of the blood. In some cases the surface was blued independent of any cardiac influence, from vaso-motor disturbance. This was so marked as at times to cause suspicion that the patient had a mild form of Reynaud's disease.[1] The nervous phenomena of influenza and its sequelæ resembled by turns every known form of neurotic disease. The force of the poison seemed to be centred in the cardiac nerves. These cases seemed to be developed as a result of the long-continued action of the influenzal poison, commencing more frequently in the late spring and early autumn; seeming to be a nervous form of influenza, some assuming this form and some running into a distinctly catarrhal fever.

Angina in several cases followed catarrhal fever, which is a further proof of the identity of the cause of the two affections.

ANGINA AND ANGINAL CASES.

In old people with angina pectoris previously, sudden death was quite common, and old gouty cases were especially liable. In the young and vigorous anginic symptoms were frequent, but death seldom, if ever, occurred. Painful attacks of the heart were very rare in old crippled hearts.

A few of the marked cases noted will be given briefly, and will illustrate the variety of cases met with; fully seventy having been seen by the writers in the last two years, most of them following immediately upon attacks of influenza or its sequelæ, but some where no such preceding attack could be clearly made out. From their close resemblance to the others it seemed fair to infer these were due to the same predisposing cause. The exciting causes were the same as those of ordinary angina,—emotion, exhaustion, indigestion, venery. The cases occurred most often preceding an unfavorable change in the weather,—on damp and murky days, with east or easterly winds.

Duration.—One case ran over three months, in which the paroxysms were broken or interrupted by catarrhal pneumonia, by asthma, and by influenzal rheumatism, these suddenly in turn replaced the angina, which on the cessation of each as suddenly returned.

Location.—The chest pain was generally on the left side, below, above, to the right, or to the left of the nipple, sometimes extending to the point of the shoulder, elbow, or even the wrist, and sometimes to the ulnar and

[1] See Case XI.

at others to the radial distribution, occasionally to both (or parts of both); often all around the arm, or in front or back, outer or inner side. A numb sensation was experienced either during the pain or before or after. Some cases started with numbness or pain at the finger-tips or wrist, from which the sensation rapidly travelled up, and merged in the breast-pain, often almost or quite as severe as "breast pang," with, in some rare cases, the feeling of impending death. Most of those manifesting fear were old angina cases and were aged people, but some few were unfamiliar with the symptoms and previously vigorous. There was sometimes the feeling that the heart was bursting; symptoms of thoracic compression and suffocation were quite common, and thus caused the patient to take short respirations and keep quiet. Sometimes the heart was accelerated; sometimes slowed. Some hearts were excitable, some not. The symptoms of angina seemed to be followed or interchanged with simple weak heart without pain. There was also often arythmia.

A few cases may be given illustrating the types of angina and anginal attacks.

Case XII.—Dr. S., aged sixty-two, in the winter of 1892 had well-marked influenza, and for the next two months he had palpitation on exertion, with intermittent pulse, irregular at times. First sound of the heart was very short and faint; no murmur. A little swelling of the feet and leg œdema. He had marked pain just above the left nipple, going to point of the left shoulder and down the front of the arm and forearm to the wrist, the thumb, index, and middle finger becoming numb and remaining so after subsidence of the pain for some hours. The symptoms yielded to iron, quinine, digitalis, and hypophosphites.

Case XIII.—Mr. J. R. S., eighteen years old, one year ago had grippe and was sick a week; four months ago he had the first palpitation; and a few days ago, after violent exercise in the gymnasium, faintness, pain over both mammary regions and across the sternum, going down the left side of the chest and down the arm to the elbow. The pain was exceedingly severe in the chest and down the arm, and greatly increased by each heart beat. Dancing and hearty meals would develop an attack. He rapidly recovered under treatment.

Case XIV.—This illustrates the association of this epidemic with gout. Mrs. M., sixty-one years old. Father had had gout, from which the fingers and toes had been swollen for many years. Seven weeks after an attack of influenza, after any exertion she would be seized with shortness of breath, pain under the costal cartilages to left of ensiform, radiating to the left shoulder and down to the elbow. She had the same symptoms after the grippe two years ago.

Case XV.—Mr. J. H., thirty-three years old, had influenza, "influenzal pneumonia," asthma, and attacks of angina for one month. This asthma with slow recovery and subsidence of anginose symptoms. The pain was excruciating, with shortness of breath, variable pulse, not rapid, subnormal, the pain going to the shoulder-point and extending down the outside of the arm to the elbow and inner aspect of the forearm to the little and ring fingers. These symptoms continued three and a half months before recovery.

Case XVI.—Dr. P., aged fifty-three, had pain at the juncture of the cartilages with the lower end of the sternum, extending through the body (apparently) to point of the left shoulder. The pain was severe, but no fear of death. The attacks lasted two or three minutes, and occurred every few days without ascertainable cause.

Case XVII. (Being one of seven cases of similar nature seen by the writers.)—

Mrs. Y., a widow for ten years, has attacks of anginic pains commencing in the clitoris, throbbing and shooting pain up to the umbilicus, left shoulder, and down the left arm, a numbness with and following the pain. Worried and annoyed but not frightened. No fear of death.

CASE XVIII.—J. T. F., aged forty-seven, had mild attacks of influenza from which she seemed perfectly recovered. On May 18 she walked in the country a mile and a half from the station to her country house and returned the same way. On reaching home was tired and went to bed early, and experienced nothing amiss until she attempted to rise in the morning, when after walking a few steps she became dizzy, had a feeling of numbness and pain beginning in both hands, running up the arms, and followed at once by intense precordial pain, violent palpitation, retching, and syncope. It was some minutes before she recovered consciousness, and any attempt to rise or sit up was followed by similar attacks, each preceded by the same feeling in the hands and arms. Food even in small quantity excited the pain, palpitation, and nausea. These attacks ended in profuse sweating. The onset was attended with a dusky flush of the face, but then left her very pale. Under rest and alcoholic stimulation the attacks became after the first day less and less violent, and during the last week of convalescence were confined to one a day, shortly after eating breakfast, after which she could sit up and even walk about a little.

CASE XIX.—C. G., aged fifty-seven, had attacks of a similar character but lighter while going about; they occurred between eleven and twelve o'clock, generally when on the street, but under treatment she recovered completely in a few weeks.

CASE XX.—L. O., aged forty-eight, had a similar history of light attacks coming on at irregular intervals, generally at a meal. Violent precordial pain, numbness in one or both arms, and at times syncope; after about three weeks she was seized with a more violent pain, fainted, and was unable after this for three weeks to rise, every attempt bringing on faintness, numbness, pain, and nausea. During this time she was unable to take food except in the smallest quantities, any attempt to give more completely demoralizing the stomach; in fact all digestive power seemed lost; even turning the patient from side to side in the bed would bring on a paroxysm, while the pulse at the wrist seemed good in apparent syncope. Temperature remained at 97° for two weeks, taken four times in twenty-four hours. Recovery was rapid after this and temperature normal; strength seemed to have completely returned.

HEART INNERVATION.

Pathology.—The generally accepted views in regard to the innervation of the heart do not prevent the supposition or hypothesis that both rapid and slow action may be produced by either excitation or partial interruption of the function of the pneumogastrics. These nerves are also concerned in ordinary angina, hence we may suspect that the poison of the disease may act as an irritant, stimulant, or sedative to this nerve, or that (and in the absence of post-mortem research we are free to theorize) the nerve itself may by inflammatory changes, due to its actual invasion by the diseased process, be partially cut off from the general distribution, a partial imperfect section, a paresis produced by disorganization. In such cases, especially if both nerves be affected, death probably occurs, preceded by irregular and tumultuous action for a few moments; while in other cases, from the evanescent symptoms, we must either conjecture that the function of the nerve is affected temporarily, or that the pathological changes are so slight in character that a return to healthy action may be easy and rapid.

The sympathetic and vaso-motor system may also play an important part in some, as it is supposed to do in many cases of ordinary angina, indeed some cases we have recorded seemed to be wholly or almost wholly due to this, as where flushing or blueness occurred paroxysmally without manifest cardiac disturbance.

Whether the irritation of the nerves was direct or reflex, whether the cardiac ganglia and the inhibitory ganglia of the heart itself were affected, are questions for further inquiry; the latter query arose after studying the cases where synchronous action was lost, or the condition known as arythmia.

Treatment.—For simple heart-failure, first in importance was alcohol, after which citrate of caffeine and cactus grandiflora were well borne by the stomach. Ether and ammonia often were not. Citrate of caffeine must be given in small dose,—one-grain doses are large enough; doses of three to five grains often produce headache and general nervous excitement. Digitalis and strophanthus were of use; atropia seemed to exercise a special influence for good. Nitro-glycerin seemed to act favorably with aged persons and those having a gouty diathesis at any age. Strychnia is often of great service. It should be given in small tonic, not in heroic, doses. In anginose cases sometimes it was found useless. If the case is anæmic it is important to build up the general system by hypophosphites, iron, strychnia, and quinine. Dyspepsia must be cured or relieved by peptonized food or by pepsin itself: in fact great care must be exercised in feeding; meat often causes recurrences; a large amount of any food was generally badly managed, and meals towards evening were badly borne. Cod-liver oil and malt benefited chronic cases.

Infrequent Heart.—Avoidance of mental worry or actual cares, or any attempt to work. Encouragement was of great use; the heart should not be much or often examined, nor any opinion unfavorable to its strength or soundness expressed. Stimulation (alcoholic) was vitally necessary.

Arsenic was valuable in anæmic cases, and one-drop doses of Fowler's solution before feeding seemed in some cases to increase retentive and digestive power. Bromide of ammonium quieted restlessness; sulphonal generally had a good effect, but was occasionally dreaded by the patient, but generally it was the most satisfactory hypnotic, often combined with bromides. Paraldehyde when well borne was useful.

The prognosis was hopeful, in almost every case, no matter how desperate it seemed, except in the aged, with organic heart-disease, cardiac degeneration, and senile weakness. Recovery was the invariable rule in the young and robust.

Treatment of the anginal cases differed but little from that employed for weak hearts from other causes. Excessive stimulation and over-stimulation of the nerves was to be guarded against, for it sometimes aggravated the symptoms. The quieting effect of rest in bed, with the attending protection from cold, fatigue, and draughts, was of the greatest importance in the treatment of the anginose cases.

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